Clinical microbiological case: a firm, right infraclavicular mass in an adult woman with connective tissue disease

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DIAGNOSIS

Subpectoralis abscess due to group B β-hemolytic streptococcus (*Streptococcus agalactiae*) (GBS).

DISCUSSION

1. The clinical presentation of GBS infections in non-pregnant adults includes primary bacteremia, followed by skin and soft tissue infection (cellulitis, surgical or burn wounds, ulcers and abscesses) [1,2,5–8]. Our patient had a subpectoral abscess and an underlying connective tissue disease. The presence of a chest wall abscess due to GBS is exceptional [1,3,4,7], and has been reported only in recent years, in one case [1]. One or more conditions predisposing to infection can be identified in most adults suffering from invasive disease (diabetes mellitus, cancer, liver disease). Connective tissue diseases are exceptional predisposing causes [1,5–8,10]. Thus, only systemic lupus erythematosus [9–11] and rheumatoid arthritis [7,9,10] have been reported as underlying medical conditions. Undifferentiated connective tissue disease has not been reported before as an underlying disease in adults with invasive GBS infection.

2. The first step in identifying this bacterium requires observation of β-hemolysis on the blood agar plate, which appears as complete clearing (lysis of erythrocytes) of the blood. While most bacteria are non-β-hemolytic, *Streptococcus* (*Streptococcus pyogenes*; *S. agalactiae*; certain streptococci included in the *S. viridans* group, such as *S. anginosus* groups and *S. mutans*; *S. dysgalactiae* subsp. *equisimilis*; *S. porcinus*; *and S. iniae*), *Staphylococcus*, *Enterococcus (Enterococcus durans)*, *Listeria monocytogenes*, *Bacillus cereus* and some Enterobacteriaceae are β-hemolytic in sheep blood agar. *B. anthracis* is non-hemolytic or very weakly hemolytic.

The second step involves recognition of the organism's morphology (coccus or rod) by Gram-staining of the colony. The catalase reaction is also helpful in distinguishing Gram-positive cocci (streptococci and enterococci are catalase negative). Small, grayish, catalase-negative, β-hemolytic colonies suggest GBS.

The third step involves identifying the β-hemolytic streptococci using traditional phenotypic criteria [Lancefield serologic grouping and the CAMP test (a lytic phenomenon produced by the synergistic action of a beta hemolysin of *S. aureus* and the CAMP factor of group B streptococci as was defined originally by christie, Atkins and Munchen-Petersen)]. In our experience, rapid antigen extraction and agglutination directly from the colonies (Slidex Strepto-Kit; BioMérieux, Durham, NC, USA) is very useful, especially for identifying groups A and B; it is less reliable for groups C, D, F and G. *Streptococcus suis*, *Streptococcus porcinus*, *Streptococcus iniae* and some *Streptococcus anginosus* groups do not agglutinate with Lancefield antibodies.

Finally, a susceptibility test with selected antibiotics helps to clarify the identity of the microorganism. In this case, the GBS was susceptible to penicillin, erythromycin, vancomycin, cefotaxime, clindamycin, and oxacillin.

3. Therapy with penicillin G for a minimum of 14 days and surgery (incision and drainage of abscesses when needed, or debridement of devitalized tissue) are recommended for the treatment of soft tissue infections due to GBS [1,12]. GBS isolates remain uniformly susceptible to penicillin, ampicillin, cephalosporins [12,13], excluding cefoxitin [12], vancomycin, teicoplanin, and carbapenems [12,13]. Some isolates with intermediate susceptibility to penicillin and cephalosporins have been reported [7,14–16], as well as some strains with vancomycin MICs up to 4 mg/L [14,16] and imipenem MICs up to 2 mg/L [14]. In our institution, since 1989, all isolates of GBS have been susceptible to penicillin, ampicillin, cephalothin, cefotaxime, and vancomycin, but erythromycin resistance increased from 5% in 1989 to 9% in 2000.

The breakpoint of in vitro susceptibility of penicillinase-resistant penicillins (PRPs), which correlates with the successful treatment of streptococcal infections, has not been determined. Although PRPs are active in vitro against streptococci, their MICs for these microorganisms are higher than the MICs of penicillin G [17]. The oxacillin MIC for GBS varies from 0.04 to 0.8 mg/L [18], and the

mean MIC is 0.06 mg/L [17], so antibacterial activity should be high enough to eradicate most streptococci. Our patient had an invasive GBS infection. The GBS MIC for oxacillin was $\leq$1 mg/L, but the patient continued to have fever and chest pain despite therapy with oral cloxacillin, and a change in therapy was needed. We do not know whether parenteral cloxacillin would have improved the response of the patient.

Although penicillin and ampicillin are the drugs of choice for the treatment of GBS infection, the in vitro activities of PRPs, cephalosporins, carbapenem and vancomycin suggest that these agents might be used for the successful treatment of serious invasive GBS infections. More studies are necessary to define fully the efficacy of agents other than ampicillin and penicillin G in the treatment of patients with invasive GBS disease.

GBS strains are uniformly resistant to co-trimoxazole and aminoglycosides [12], but the latter can be used as synergistic therapy with penicillin for the treatment of endocarditis [12] and infection caused by a penicillin-tolerant microorganism [14]. The clinical importance of this phenomenon is unknown [12,14].

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